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Case Report

Pneumomediastinum in interstitial lung disease-A case report

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ABSTRACT

Pneumomediastinum (PM) is a relatively rare disease and is defined as changes in pressure gradients within the thoracic cavity secondary to increases in intrathoracic pressure which ultimately leads to rupture of alveoli and dissection of air along the fascial planes of the tracheobronchial tree. PM differs from secondary pneumomediastinum in that the latter, by definition, requires a pathologic etiology. Although the presence of secondary pneumomediastinum on radiographic imaging may be the result of significantly serious events such as chest trauma, esophageal rupture, or infection with gas producing organisms, the natural history of PM tends to be benign and self-limiting, resolving over approximately one week. In this report we describe a case of a patient with underlying pulmonary fibrosis presenting with persistent dyspnea found to have pneumomediastinum on computed tomography (CT) that resolved completely over a 3 day period with the use of high concentration oxygen therapy.

1. Case report

A 60 year old woman with history of interstitial lung disease presented with a 2 week history of escalating dyspnea characterized by difficulty with activities of daily living and hypoxia at rest. Two months prior, she was noted to be hypoxic on ambulation with saturations of 80% with exertion and was started on oxygen with sleep and ambulation. On this presentation, her oxygen saturations were 88% on room air with nocturnal oxygen saturations less than 85%. A CT of the chest was performed and demonstrated worsening pulmonary fibrosis with honeycombing, traction bronchiectasis, subpleural bullae, and significant mediastinal emphysema tracking from the diaphragm to the neck (Fig. 1). The patient initially refused hospital admission and remained at home. Over the subsequent 4 days, she developed progressive dyspnea and was admitted to the hospital. On admission, her oxygen saturation was 74% on room air. Creptius over the anterior chest and neck were absent as was the classic "Hamman's sign" defined as crepitus that is synchronized with the hearbeat.[1] Laboratory values were notable for a leukocytosis of 17,000 K/uL, an elevated bicarbonate level of 35, with a pH of 7.51, $PaCO_2$ of 54 mmHg, and a paO_2 of 72 mm Hg on oxygen. A repeat CT scan of the chest revealed no significant changes in the mediastinal emphysema from the images 4 days prior (Fig. 2 and 3).

2. Diagnosis

Secondary pneumomediastinum due to bleb rupture.

3. Clinical discussion

Pneumomediastinum is the presence of air in the mediastinum. It was first described in 1939 by Louis Hamman and hence the associated "Hamman Crunch".

In the current case we attribute the presence of symptomatic pneumomediastinum to bleb rupture. Associated lung disease may adversely affect the outcome of pneumomediastinum.

The clinical presentations of pneumomediastinum are varied and often complex. Classically, these include symptoms of dyspnea, odynophagia, voice hoarseness, and retrosternal chest pain. In various studies performed, the leading cause of symptomatic pneumomediastinum was found to be chest pain. Pneumomediastinum is often complicated by pneumopericardium and pneumothorax due to air tracking along the bronchovascular bundle and allowing for an increase in the compartmental pressure while decreasing the venous return and cardiac output which may present as a medical emergency. Pneumopericardium usually presents as increasing dyspnea, cyanosis, engorgement of neck veins, thready rapid pulse, and hypotension. Treatment for severe symptomatic PM may include surgically placed subcutaneous "venting" incisions where the air has accumulated.[2]

In this case report a patient with underlying pulmonary fibrosis presented with persistent dyspnea.

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Fig. 1. CT revealing air tracking around the bronchovascular bundle and pericardium along with honeycombing.

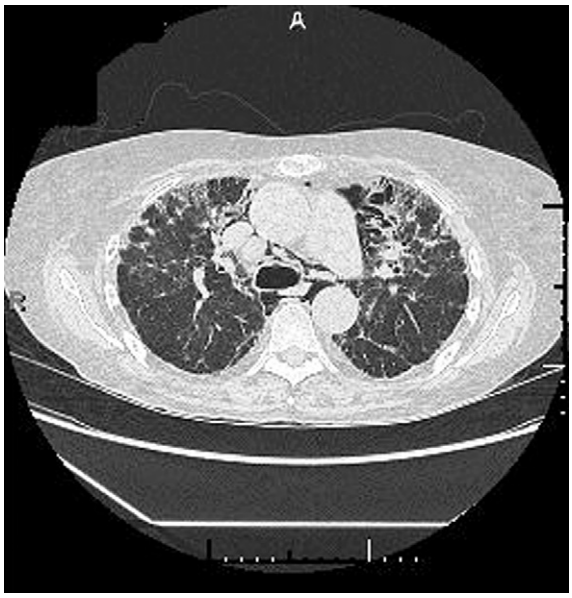

Fig. 2. Unchanged CT scan on admission to the hospital.

4. Radiological discussion

The diagnostic studies performed to evaluate patients with pneumomediastinum begin with a chest X-ray which has been the sole test in some studies. CT scan has become the gold standard to diagnose pneumomediastinum. Studies reveal that chest radiography may miss up to 30% of pneumomediastinal cases when compared to that of CT, as was evident in this case. Other imaging modalities that are done to rule out secondary causes of pneumomediastinum are contrast eosophagogram, esophagoscopy, fiber optic bronchoscopy, and CT scans of the abdomen.[3,4]

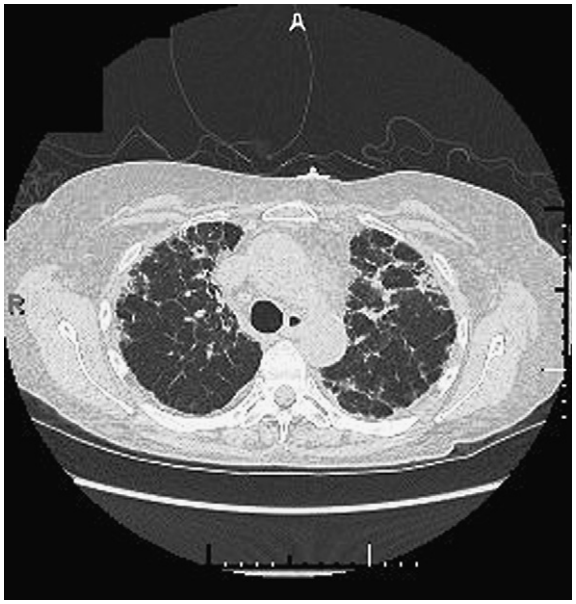

Fig. 3. CT scan showing resorption of air after 3 days of treatment with high concentration oxygen therapy.

5. Conclusion

Pneumomediastinum is usually a self-limiting, benign process treated with bed rest and analgesics lasting approximately 2 weeks in the majority of cases. Given the severe exacerbation of dyspnea and progressive hypoxia in the case of our patient, we treated her with a high concentration of oxygen. Although there is not an abundance of data regarding the mechanism of resolution of the pneumomediastinum due to high flow oxygen therapy, some studies suggest that, by breathing 100% oxygen, Nitrogen is dissipated from the blood, increasing the gas absorption gradient.[5]

Pneumomediastinum is usually a benign condition that can be treated on an outpatient basis, but the presence of additional lung disease may warrant admission to the hospital. It is important to consider pneumomediastinum as a potential cause of shortness of breath in patients with history of lung disease, particularly interstitial lung disease or the presence of blebs to prevent complications. It is important to have a high suspicion of pneumomediastinum as it could be easily missed.

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